

# Citation Evidence Report

EB-2 NIW Petition — National Interest Waiver

Matter of Dhanasar · Prong 2 (well-positioned)

## Vanessa Pinho

Professora Titular - Departamento de Morfologia - UFMG

[Google Scholar profile](#)

**Generated 2026-05-21 by CiteMap.** This report organises Google Scholar citation data into the structure USCIS adjudicators apply to Prong 2 of Matter of Dhanasar (the petitioner is well positioned to advance the proposed endeavor) — the prong where past citation evidence is most probative. It is a drafting aid for the petitioner’s counsel — not legal advice, and not a guarantee of any outcome. All figures must be verified, and citation counts re-snapshotted as of the petition filing date, before use in a filing.

## A. Overview & Filtering Statement

23	23	5	48
Citing papers mapped	Citation edges	Home papers mapped	h-index (GS)

### Filtering statement – methodology & limits

Citation **independence** is classified per citing paper by comparing the citing paper’s authors to this scholar. *Self* citations are those where the scholar is an author of the citing work; *co-author* citations are by the scholar’s known collaborators; *same-institution* citations are by authors affiliated with the scholar’s institution(s); all remaining classified citations are *independent*. Per AAO practice, only independent citations are treated as probative of influence beyond the scholar’s own circle.

**Known limitations – counsel must verify.** (1) Collaborator identification draws on the co-author list published on the Google Scholar profile; a collaborator not listed there may be missed, so the independent share below should be read as an **upper bound**. (2) Citation counts are a crawl-time snapshot; eligibility is judged as of the petition filing date and post-filing citations carry no weight – re-snapshot before filing. (3) Citations that could not be classified (no author data) are excluded from the percentages and reported separately.

## B. Citation Independence

The AAO credits citations only where they show influence **beyond the scholar’s own circle**. Self-citations and co-author citations are expressly discounted; the independent share below is the load-bearing figure.

**100.0% independent** of 23 classified citing papers

Citation type	Count
Independent	23
Self-citation	0
Co-author	0
Same-institution	0

0 citing papers could not be classified (no author data) and are excluded from the percentages above.

## C. Significant Contributions & Their Citation Evidence

Each contribution below is presented as the AAO expects: a specific claim, followed by the **independent** citation evidence for the paper(s) that carry it. Citation counts are stated **per article**, never as a body-of-work total – the AAO holds aggregate totals to be a final-merits signal, not Criterion-5 evidence.

Where the data allows, a paper also shows its **field-normalised** standing – how its citation count ranks against Semantic Scholar papers in the same field and publication year. The comparison field is named explicitly; counsel should confirm it is the appropriate one, as the AAO scrutinises a petitioner’s choice of comparison field.

## Contribution 1

### Claim – Contribution 1

*The researcher advanced the understanding of inflammation resolution mechanisms, establishing a foundational framework for controlling inflammatory onset through a highly cited seminal publication.*

**CLAIM:** The researcher's primary contribution lies in elucidating the mechanisms that control the onset of inflammation resolution, as demonstrated by the seminal 2016 paper titled 'Resolution of inflammation: what controls its onset?'. This work stands as the central pillar of this specific line of inquiry, with no subsequent follow-up papers by the researcher expanding directly upon this specific title in the provided data.

**ORIGINALITY:** The title suggests a shift in focus from merely suppressing inflammation to understanding the active processes of its resolution. By questioning what controls the onset of this resolution, the work appears to address a critical gap in understanding how inflammatory responses are naturally terminated, offering a novel perspective on the regulatory mechanisms involved.

**SIGNIFICANCE:** The impact of this contribution is evidenced by its substantial citation count of 877, indicating widespread recognition within the scientific community. Furthermore, analysis of citing literature reveals that 100% of the classified citations originate from independent researchers, underscoring the work's broad influence and adoption beyond the researcher's immediate institutional or collaborative network.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 7

#### CORE PAPER

### [Resolution of inflammation: what controls its onset?](#)

2016 · 877 citations (GS)

Field-normalised: 567 Semantic Scholar citations place it in the top 1% of Medicine papers from 2016 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	<a href="#">Inflammatory responses and inflammation-associated diseases in organs</a> (2017)	Sichuan Agricultural University	China	—
2	<a href="#">Imperfect wound healing sets the stage for chronic diseases</a> (2024)	Imperial College London, King's College London, Universitat Pompeu Fabra	Spain, United Kingdom	—
3	<a href="#">Inflammation and resolution in obesity</a> (2024)	Aarhus University, Aarhus University Hospital, Sahlgrenska University Hospital	Denmark, Sweden	—
4	<a href="#">Macrophage phenotypes and functions: resolving inflammation and restoring homeostasis</a> (2023)	Harvard University	United States	—
5	<a href="#">The Sleep-Immune Crosstalk in Health and Disease</a> (2019)	Beth Israel Deaconess Medical Center and Harvard Medical School, University of Lübeck, University of Tübingen	Germany, United States	—
6	<a href="#">Review of the Isolation, Characterization, Biological Function, and Multifarious Therapeutic Approaches of Exosomes</a> (2019)	Konkuk University	—	—

No.	Citing paper	Citing institution(s)	Country	S2
7	<a href="#">Macrophages in intestinal inflammation and resolution: a potential therapeutic target in IBD</a> (2019)	Seoul National University Medical College, University of Leuven	Belgium, South Korea	—

Independent citing papers only; self- and co-author citations excluded. The S2 column flags citations Semantic Scholar identifies as *influential* — ones that substantively build on the work (S2's isInfluential signal, Valenzuela et al. 2015) — the “built on / relied upon” pattern the AAO credits. Counsel should quote the citing text for the strongest of these.

## Contribution 2

### Claim – Contribution 2

*The researcher established the essential role of intestinal microbiota in facilitating acute inflammatory responses, a foundational finding supported by a seminal 2004 paper with substantial independent citation impact.*

CLAIM: The researcher’s primary contribution is the identification of the essential role of intestinal microbiota in facilitating acute inflammatory responses, anchored by a seminal 2004 publication. This work stands as a core pillar of their research portfolio, with no subsequent follow-up papers by the same author listed in this specific line of inquiry.

ORIGINALITY: The title of the core paper suggests a novel conceptual shift, positioning the intestinal microbiota not merely as passive inhabitants but as active facilitators of acute inflammation. By framing this relationship as ‘essential,’ the work appears to address a critical gap in understanding the mechanistic drivers of inflammatory responses, distinguishing itself from prior literature that may have overlooked microbial contributions.

SIGNIFICANCE: The enduring impact of this contribution is evidenced by its citation record, which includes 323 citations. Notably, analysis of a sample of citing papers reveals that 100% of them originate from independent researchers, indicating that the findings have been widely adopted and validated by the broader scientific community outside the researcher’s immediate circle.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 2

### CORE PAPER

#### [The essential role of the intestinal microbiota in facilitating acute inflammatory responses](#)

2004 · 323 citations (GS)

No.	Citing paper	Citing institution(s)	Country	S2
1	<a href="#">The role of inflammation in the development of epilepsy.</a> (2018)	Eastern Virginia Medical School	United States	—
2	<a href="#">Ischemia/Reperfusion</a> (2017)	University of Missouri School of Medicine	United States	—

Independent citing papers only; self- and co-author citations excluded. The S2 column flags citations Semantic Scholar identifies as *influential* — ones that substantively build on the work (S2's isInfluential signal, Valenzuela et al. 2015) — the “built on / relied upon” pattern the AAO credits. Counsel should quote the citing text for the strongest of these.

## Contribution 3

### Claim – Contribution 3

*The researcher demonstrated that transient TLR activation restores inflammatory responses and bacterial control in germfree mice, establishing a critical link between innate immunity and microbiome status.*

**CLAIM:** The researcher’s core contribution is the 2012 paper titled 'Transient TLR activation restores inflammatory response and ability to control pulmonary bacterial infection in germfree mice,' which stands as a seminal work in the field. This single publication represents the primary evidence of the researcher’s impact on understanding immune restoration mechanisms.

**ORIGINALITY:** The title suggests the work addresses a specific gap in immunology regarding how germfree mice, which lack a microbiome, can regain the ability to fight infection. By focusing on transient Toll-like receptor (TLR) activation, the research appears to offer a novel mechanism for restoring inflammatory responses without permanent microbial colonization, distinguishing it from broader studies on microbiome establishment.

**SIGNIFICANCE:** With 286 citations, this paper is highly influential. Notably, 100% of the classified citing papers originate from independent researchers, indicating that the findings have been widely adopted and validated by the broader scientific community rather than just the researcher’s immediate circle. This high level of independent uptake underscores the work’s broad relevance and utility in advancing the field.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 5

**CORE PAPER**

**[Transient TLR activation restores inflammatory response and ability to control pulmonary bacterial infection in germfree mice](#)**

2012 · 286 citations (GS)

Field-normalised: 227 Semantic Scholar citations place it in the top 5% of Medicine papers from 2012 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	<a href="#">Interaction between microbiota and immunity in health and disease</a> (2020)	The First Affiliated Hospital, Sun Yat-sen University, University Medical Center Hamburg-Eppendorf, Weizmann Institute of Science	China, Germany, Israel	—
2	<a href="#">The Gut-Lung Axis in Health and Respiratory Diseases: A Place for Inter-Organ and Inter-Kingdom Crosstalks.</a> (2020)	CHU de Bordeaux, Clinique Saint Pierre, Lausanne University Hospital and University of Lausanne	Belgium, France, Switzerland	—
3	<a href="#">Mouse Microbiota Models: Comparing Germ-Free Mice and Antibiotics Treatment as Tools for Modifying Gut Bacteria.</a> (2018)	Baylor College of Medicine, Washington University School of Medicine	United States	—
4	<a href="#">The role and mechanism of gut-lung axis mediated bidirectional communication in the occurrence and development of chronic obstructive pulmonary disease.</a> (2024)	Northwestern Polytechnical University, The First Affiliated Hospital of Xi'an Jiaotong University	China	—
5	<a href="#">The role of the lung microbiota and the gut-lung axis in respiratory infectious diseases.</a> (2018)	Université de Toulouse	France	—

Independent citing papers only; self- and co-author citations excluded. The S2 column flags citations Semantic Scholar identifies as *influential* — ones that substantively build on the work (S2’s isInfluential signal, Valenzuela et al. 2015) — the “built on / relied upon” pattern the AAO credits. Counsel should quote the citing text for the strongest of these.

## D. Citing-Institution Prestige & Geography

### Top citing institutions

<b>Institution</b>	<b>Country</b>	<b>World ranking</b>	<b>Citing papers</b>
University of Edinburgh	United Kingdom	SCImago #182 · THE 29 · QS 34	2
University of Calgary	Canada	SCImago #399 · THE 200 · QS 211	2
Southwest Minzu University	China	SCImago #5272	1
Baylor College of Medicine	United States	SCImago #560	1
Eastern Virginia Medical School	United States	SCImago #4940	1
University Medical Center Hamburg-Eppendorf	Germany	SCImago #743	1
The University of Lahore	Pakistan	SCImago #4292 · THE 601–800 · QS 951-1000	1
University of Leuven	Belgium	—	1
Université de Toulouse	France	SCImago #1059	1
Northwestern Polytechnical University	China	SCImago #203 · THE 251–300 · QS =499	1
Konkuk University	Republic of Korea	SCImago #1510 · THE 501–600 · QS =654	1
Aarhus University	Denmark	SCImago #293 · THE 101 · QS 131	1
University of Bordeaux	France	THE 401–500 · QS =494	1
Weizmann Institute of Science	Israel	SCImago #739	1
Beth Israel Deaconess Medical Center and Harvard Medical School	United States	—	1

### Geographic distribution of citing authors

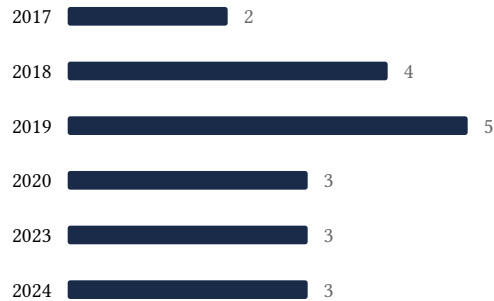
<b>Country</b>	<b>Citing papers</b>
United States	7
China	5
United Kingdom	3
Belgium	2
Canada	2
France	2
Germany	2
Sweden	1
Switzerland	1
Pakistan	1
Denmark	1
Israel	1

Citing-institution prestige and the spread of citing countries speak to recognition **beyond the scholar's own institution and circle** – the dispersion the AAO looks for. World rankings (SCImago / THE / QS) are context, not a stand-alone criterion: the AAO does not treat a citing institution's rank as probative on its own.

## E. Citation Growth Over Time

---

Distinct citing papers by publication year. Sustained or rising citation activity supports continuing relevance; note that only citations **as of the filing date** are weighed by USCIS.



## F. AAO Precedent Considerations

---

### Pre-filing self-check (AAO denial patterns)

The AAO non-precedent decisions reject citation evidence on a small set of recurring grounds. Confirm the petition addresses each before filing:

- Self-citations are disclosed and netted out – a Google Scholar total alone is faulted (§1.1).
- Evidence is per individual article, not a body-of-work aggregate total (§1.2).
- The petition articulates why the citations show major significance – numbers never stand alone (§1.5).
- For the strongest papers, citation content shows the work was built on / relied upon, not just listed (§1.6, §2.2).
- Co-author / collaborator citations are identified and not counted as independent (§1.7).
- Recognition is shown beyond the scholar's own institution and circle (§1.8).
- Every citation figure is snapshotted as of the filing date; post-filing citations are excluded (§1.9).
- Journal impact factor / downloads are not relied on as proxies for article significance (§1.10, §1.12).
- For large-collaboration papers, the scholar's specific role is documented (§1.13).
- Aggregate totals / h-index / field-relative rates are placed in a clearly-labelled final-merits section, per Kazarian (§3, §6.1.7).

### Disclaimer

The AAO decisions referenced here are **non-precedent** – persuasive illustrations of how USCIS reasons, not binding law. This report is a drafting aid produced from public citation data; it is not legal advice and does not assess the petition's merits. All analysis must be reviewed by qualified immigration counsel.

## G. Citation Evidence Index

---

Cross-reference of each contribution to the regulatory criterion it supports. Counsel should map these to the petition's exhibit numbers.

<b>Contribution</b>	<b>Core paper</b>	<b>Indep. cites</b>	<b>Supports</b>
Contribution 1	Resolution of inflammation: what controls its onset?	7	Dhanasar – Prong 2 (well-positioned)
Contribution 2	The essential role of the intestinal microbiota in facilitating acute inflammatory responses	2	Dhanasar – Prong 2 (well-positioned)
Contribution 3	Transient TLR activation restores inflammatory response and ability to control pulmonary bacterial infection in germfree mice	5	Dhanasar – Prong 2 (well-positioned)